DISSERTATION ON

THE STUDY ON

EFFECT OF FOAM SCLEROTHERAPY IN VARIOUS VASCULAR AND LYMPHATIC MALFORMATIONS

This dissertation is submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfillment of the requirement of the award
for the degree of

M.D BRANCH XII

DERMATOLOGY, VENEREOLOGY & LEPROSY

STANLEY MEDICAL COLLEGE,
CHENNAI – 600001
MARCH 2011

CERTIFICATE

This is to certify that this dissertation entitled **'EFFECT OF FOAM SCLEROTHERAPY IN VARIOUS VASCULAR AND LYMPHATIC MALFORMATIONS'** submitted by **Dr. B.K.AARTHI** to **The Tamilnadu Dr.M.G.R. Medical University, Chennai** in partial fulfillment of the requirement of the award for the degree of **M.D BRANCH XII (DERMATOLOGY, VENEREOLOGY & LEPROSY)** and is a bonafide work carried out by her under direct supervision and guidance.

Signature of the Head of the Department

Signature of the Dean

DECLARATION

I solemnly declare that the dissertation titled, **'EFFECT OF FOAM SCLEROTHERAPY IN VARIOUS VASCULAR AND LYMPHATIC MALFORMATIONS'** was done by me at Stanley Medical College and Hospital during 2008 – 2011 under the guidance and supervision of my Chief **Prof. Dr.K.Manoharan,M.D., D.D**

The dissertation is submitted to **THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY** towards the partial fulfillment of requirement for the award of **M.D. Degree (Branch XII) in DERMATOLOGY, VENEREOLOGY & LEPROSY**

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Place:

Date:

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CONTENTS

INTRODUCTION

INTRODUCTION

Sclerotherapy is an intravascular injection of a chemical irritant to cause chemical thrombophlebitis. The term was coined in the 1940's by American physician H. I. Biegeleisen[1]. Sclerotherapy is being used in treating varicose veins, hemangiomas and lymphangiomas. Introduced more than 150 years ago[2], sclerotherapy remains the most effective procedure for permanent eradication of pathologically enlarged as well as cosmetically undesirable but otherwise normal veins.

Innovative devices have been introduced within the past 10 years that utilize electromagnetic energy sources to target vein wall constituents directly or indirectly and either transcutaneously or intravascularly, but these procedures are significantly less effective and reproducible than sclerotherapy for permanently eradicating unwanted veins.

Foam sclerotherapy, in which the sclerosant is mixed with air or physiological gases, is more efficacious than direct injection of sclerosants[3], as air in the foam prolongs the contact of the agent with the endothelium. Hence, maximum sclerosant action can be obtained at lesser concentration and quantity. Foamed agent exerts its effect at microcirculation, which is inaccessible to other procedures.

AIM OF THE STUDY

AIM OF THE STUDY

- To assess the effectiveness of foam sclerotherapy in various vascular and lymphatic malformations.

- To observe the adverse effects of foam sclerotherapy during and after the procedure.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

VARICOSE VEINS

Varicose veins literally mean grossly dilated, tortuous elongated veins. It can occur at different places in the body. The commonest is the varicosity of the lower limbs

Etiology

The cause of primary varicose veins is incompetent venous valves that result in venous hypertension. Secondary varicose veins result from deep venous thrombosis and its sequelae or congenital anatomic abnormalities. The etiology of these varicose veins can be classified into the following three groups:

- Primary: Valvular insufficiency of the superficial veins, most commonly at the saphenofemoral junction.
- Secondary
    - Mainly caused by deep vein thrombosis (DVT) that leads to chronic deep venous obstruction or valvular insufficiency. Long-term clinical sequelae from this have been called the postthrombotic syndrome.

        - Catheter-associated deep vein thromboses are also included.
        - Pregnancy-induced and progesterone-induced venous wall and valve weakness worsened by expanded circulating blood volume and enlarged uterus compresses the inferior vena cava and venous return from the lower extremities.
        - Intra-abdominal pressure / pelvic compression
        - Trauma
- Congenital: This includes any venous malformations. A few examples are listed as follows:
    - Klippel-Trenaunay variants
    - Avalvulia

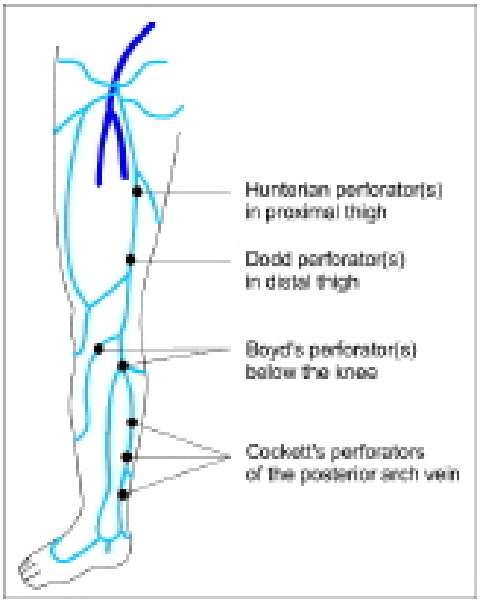
Pathophysiology

Varicose veins are simply dilated, tortuous veins of the subcutaneous/superficial venous system. However, the pathophysiology behind their formation is complicated and involves the concept of ambulatory venous hypertension. To understand this, the anatomy of the lower extremity venous system must be briefly discussed. Two venous systems are found in the lower extremity, the deep and superficial. The deep system ultimately leads backs to the inferior vena cava, then to the heart. The superficial system is found above the deep fascia of the lower extremity,

within the subcutaneous tissue. Many superficial veins exist, but they all drain into the 2 largest, the greater saphenous vein (GSV) and the short saphenous vein (SSV), formerly called the lesser saphenous vein.

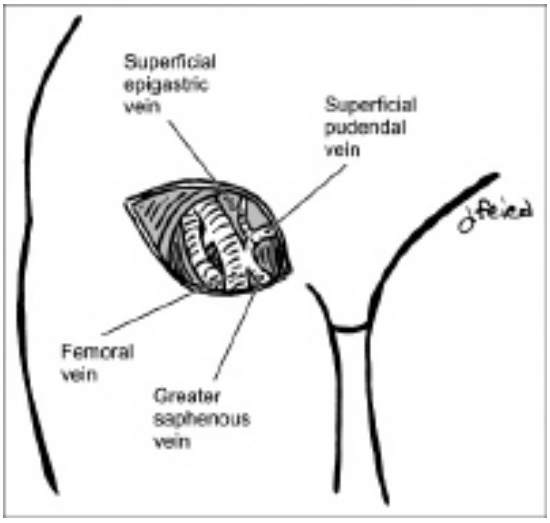
The superficial venous system is connected to the deep system at a number of the following locations:

- Perforator veins: These veins transverse the deep fascia of the lower extremity. A number of named perforators are found at the thigh, knee, and leg.



Named perforators along the greater saphenous distribution.

- Saphenofemoral junction (SFJ): This is located proximally at the groin where the GSV meets the femoral vein.



Saphenofemoral junction.

- Saphenopopliteal junction (SPJ): This is located behind the knee where the SSV joins with the popliteal vein.

In healthy veins, the flow of venous blood is through the superficial system into the deep and up the leg and toward the heart. One-way venous valves are found in both systems and the perforating veins. Incompetence in any of these valves can lead to a disruption in the unidirectional flow of blood toward the heart and result in ambulatory venous hypertension. Furthermore, incompetence in one system can often lead to incompetence in another.

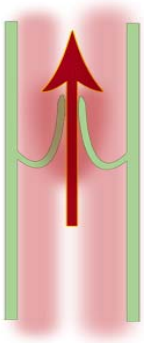
Incompetence in the superficial venous system alone usually results from failure at valves located at the SFJ and SPJ. The gravitational weight of the column of blood along the length of the vein creates hydrostatic pressure,

which is worse at the more distal aspect of the length of vein.

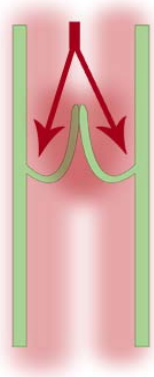
Incompetence of the perforating veins leads to hydrodynamic pressure. The calf pump mechanism helps to empty the deep venous system, but if perforating vein valves fail, then the pressure generated in the deep venous system by the calf pump mechanism are transmitted into the superficial system via the incompetent perforating veins.

Once venous hypertension is present, the venous dysfunction continues to worsen through a vicious cycle. Pooled blood and venous hypertension leads to venous dilatation, which then causes greater valvular insufficiency. Over time, with more local dilatation, other adjacent valves sequentially fail, and after a series of valves has failed, the entire superficial venous system is incompetent. As mentioned before, this can then cause subsequent perforator and deep venous valvular dysfunction.

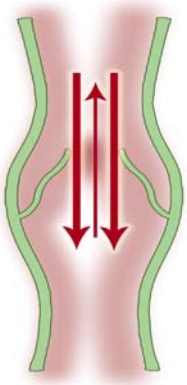
VALVES IN THE VEINS



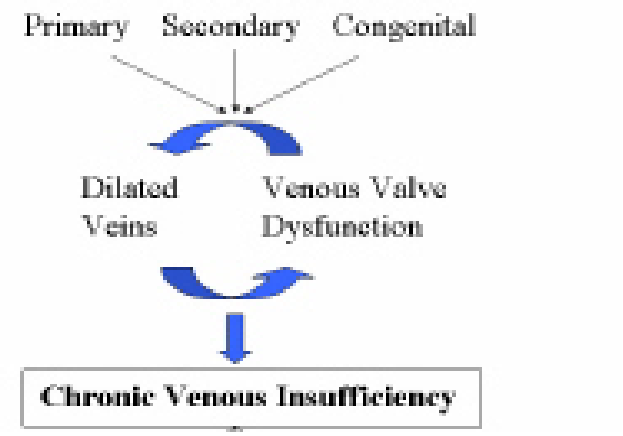
Normal vein with unidirectional valve



Unidirectional valve (Prevents backward flow)



Varicose vein with damaged valves



Pathway leading to varicose veins

The inciting etiology of superficial valvular insufficiency is often difficult to determine because the clinical manifestations of venous hypertension are delayed. The original cause can be classified as primary, secondary, and congenital as previously described.

The clinical finding of varicose veins, reticular veins, and telangiectasias are due to the hypertension in the superficial venous system that spreads to collateral veins and tributary veins, causing dilated tortuous structures. Treatment modalities are geared towards correcting the superficial venous hypertension [4]. In contrast to the superficial veins, the deep veins do not become excessively distended. They can withstand the increased pressure because of their construction and the confining fascia.

Predisposing factors

Age: As age advances the frequency of the disease increases

Sex: Most commonly seen the female population

Racial

Hereditary Factors

Hormonal: Female hormone

Pregnancy: Pressure of gravid uterus on the major veins in the abdomen

Secondary to Diseases: Fibroid Uterus, Lower abdominal tumors

Secondary to Deep Vein Thrombosis

Chronic Constipation

Occupational: Standing for prolonged period

Symptoms :

Pain: Dull aching pain in the lower leg is the commonest early symptom

Swelling of the feet: Oedema of the feet especially on standing for long time or by evening is the next common symptom.

Bleeding

Itching

Discoloration: Blackish discoloration around the ankle joints and lower leg

Eczematous Change around the ankle and foot

Lipodermatosclerosis: Thickening of the skin with depigmentation or hyper pigmentation

Venous ulcers: Initially small ulcers develop and heal spontaneously over a period of time. Later small ulcers refuse to heal and proceed to progressive skin erosion resulting in very large non healing ulcers

Investigations:

In addition to the routine investigations, the specific investigation is a Color Doppler Scan of the Lower Limb Venous System. The main purpose is to rule out any deep venous thrombosis.

Treatment of Varicose Veins:

There are different modalities of treatment for varicose veins:

1) Surgery:

i. This is the conventional treatment directed towards the ablation of the diseased veins by dissection i.e., Ligation of the feeding vessels at the inguinal region followed by Stripping of the superficial vein.
ii. Multiple subfascial ligation by different techniques/invasive as well as minimally invasive techniques.

iii. Valvoplasty: Reconstructing artificial valves in the veins, a very tedious as well as expensive procedure.

2) Minimally invasive Procedures

(a) Electrodessication:

This is the process of passing electric current through a probe to destroy the vessel wall but is rarely used today because of the disfiguring cutaneous injury.

(b) Radiofrequency ablation

Thermal energy is delivered directly to the vessel wall, causing protein denaturation, collagenous contraction, and immediate closure of the vessel. In contrast to laser therapy, the RF catheter actually comes into contact with the lumen walls.

(c) Endo venous Laser Ablation (ELT)

A laser fiber produces endoluminal heat that destroys the vascular endothelium.

(d) Sclerotherapy:

The oldest, simplest and the cheapest method of treating varicose veins, the most accepted and promising method of treatment by constant innovations in the technique. It takes hardly 10 to 15 minutes for the completion of the procedure.

HEMANGIOMAS AND LYMPHANGIOMAS :

Vascular tumors and malformations have often been described using a classification system based on morphologic characteristics. This practice has given rise to a countless number of names that are interchangeably used, often befuddling medical professionals. In 1982, Mulliken and Glowacki proposed a biologic classification of vascular tumors and malformations that has since gained wide acceptance [5]. This system correlates histologic features with historical and physical findings to provide a simplified classification of vascular lesions. The system has been modified slightly over the years to incorporate new information.

Mulliken and Glowacki use the following criteria to separate vascular malformations into 2 broad categories:

- Vascular tumors are also known as hemangiomas. These tumors exhibit endothelial hyperplasia. Approximately 30% are visible at birth, and the remaining 70% appear in children aged 2-4 years. Postnatal growth is rapid, and involution is slow. In 1996, the International Society for the Study of Vascular Anomalies added the rapidly involuting congenital hemangioma, non-involuting congenital

hemangioma, Kaposiform hemangioendothelioma, tufted angioma, and pyogenic granuloma to the list of vascular tumors.

- Vascular malformations are subdivided into high-flow (arterial, arteriovenous) malformations and slow-flow (venous, capillary, lymphatic) malformations. They exhibit normal endothelial turnover. Approximately 90% are recognized at birth. They increase in size as the child grows.

Frequency

Vascular tumors (i.e., hemangiomas) are the most common tumors in infants. They are apparent in 1-2.6% of neonates at birth across all races, according to one series. Approximately 30% of hemangiomas are recognized in the newborn nursery. Prevalence is increased in preterm infants that weigh less than 1000 g and in white children younger than 1 year. The male-to-female ratio is 3:1

The incidence rate of lymphatic malformations is 1.2-2.8 per 1000 live births [6]. Approximately 50% of lymphatic malformations are apparent at birth; 90% appear before age 2 years. Most reports indicate an equal male-to-female distribution.

Etiology

Vascular tumors (hemangiomas) are believed to result from developmental errors that occur at 4-10 weeks' gestation. Most cases are sporadic; however, they are occasionally inherited in an autosomal dominant fashion with moderate-to-high rates of penetrance [7].

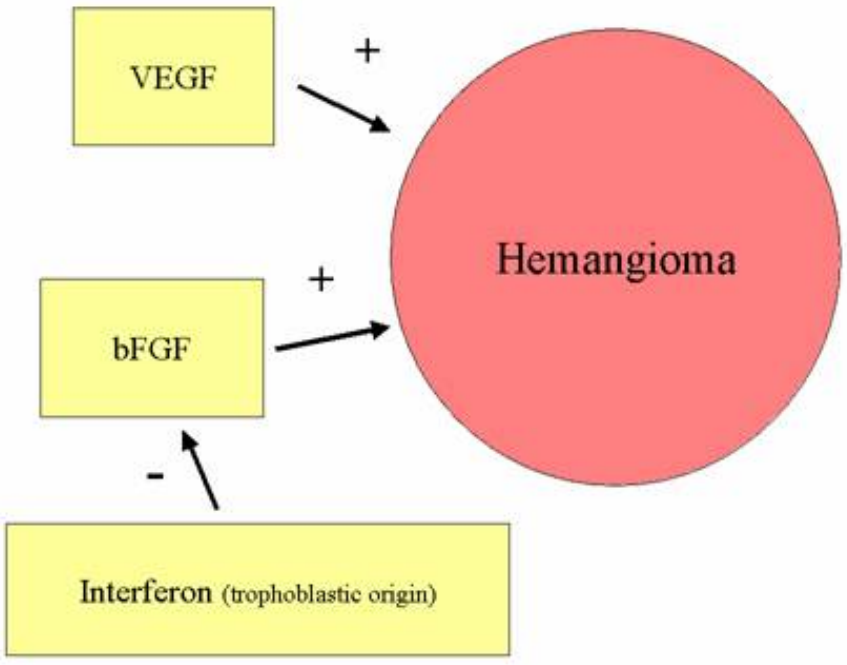
Capillary malformations are generally considered to be sporadic lesions; however, pedigrees of autosomal dominant inheritance have been reported. Port-wine stains are also associated with Klippel-Trenaunay-Weber and Sturge-Weber syndromes. Mutations in the *RASA1* gene may underlie the capillary malformation–arteriovenous malformation syndrome. A novel mutation in *RASA1* has been reported to cause capillary malformation and limb enlargement [8]. An extensive degree of phenotypic heterogeneity may be associated with deleterious mutations in *RASA1*.

The exact cause of lymphangioma formation is unknown, but most cases are believed to be sporadic. The formation of lymphangiomas possibly reflects a failure of lymph ducts to connect with the venous system during embryogenesis, abnormal sequestration of lymphatic structures, or both. Ongoing research has elucidated some of the vascular growth factors that

may be involved in formation of lymphatic malformations such as VEGF-C and flt-4. Cases secondary to trauma and infection have also been reported.

Pathophysiology

Hemangiomas are the result of abnormal changes in angiogenesis that allow the overproliferation of vascular entities. Several authors have elucidated the complex interplay of angiogenic and angiostatic forces involved in normal and pathologic processes [9]. However, fetal vascular development remains poorly understood. Many of the angiogenic markers (i.e., FGF, VEGF, E-selectin, type IV collagenase) are increased during the proliferative phase [10].



During the involutional phase of hemangiomas, a subsequent decrease in angiogenic factors occurs, with a 5-fold increase in endothelial cell apoptosis. These alterations in angiogenic factors may account for the increased vascular proliferation that occurs in hemangiomas. Mesenchymal progenitor cells may be key to infantile hemangioma development [11]. These hemangiomas may be caused by an abnormal or delayed differentiation of mesodermal progenitor cells into the disorganized mass of blood vessels.

Microscopic hemangioma tissue reveals proliferating endothelial cells. During involution, endothelial cells flatten, the vessel lumens dilate, and fibrous tissue is deposited. Recent studies have also discovered hemangioma-specific antigens not found in normal skin. These include GLUT1, merosin, and Lewis Y antigen.

Capillary malformations (port-wine stains) are groups of tortuous blood vessels located in the upper layers of the dermis. One study revealed that these lesions have decreased innervation in perivascular regions, which generates the hypothesis that the lesions are secondary to impaired vascular tone.

Lymphangiomas are collections of lymph vessels filled with serous fluid. Their histology ranges from capillary-sized vessels to macroscopic

fluid–filled vessels. Lymphatic malformations can be associated with gross anatomic deformities with severe involvement of the surrounding structures on the face or extremities. Research has begun to reveal some of the cell signals that may be involved in the formation of lymphangiomas. For example, VEGF-C has been found to be adequate in causing lymphatic hyperplasia. Evaluation of VEGF-C and VEGFR-3 in a series of lymphangiomas has suggested that superficial lymphangiomas likely represent peripheral lymphatic dilatation [12].

Presentation

Hemangiomas are most commonly located on the head and neck (59%), followed by the trunk (24%), lower extremities (10%), and upper extremities (7%) [13]. Most are less than 2 cm in diameter but, in some instances, can cover large portions of the body. Typical presenting symptoms occur superficially, and the appearance can vary from a hypopigmented macule to a bruiselike macule [14]. The course of these lesions includes a proliferative postnatal growth phase that lasts for 3-9 months, with a gradual involution that occurs over 2-6 years. Involution is usually complete by age 7-10 years. Only 50% of patients have completely normal-appearing skin at this time.

Recognizing atypical presentations and sub classifications of hemangiomas is critical for prognostic and treatment purposes. A congenital hemangioma is a lesion that is fully developed at birth. Deep hemangiomas have a blue discoloration because of their proliferation in the dermis and subcutaneous tissues. Multiple hemangiomas, often referred to as hemangiomatosis, appear as multiple smaller lesions. Pseudoclubbing may be the first sign of an underlying subungual hemangioma.

Because hemangiomas may be a part of a syndromic complex, query about other symptoms that may be associated with Klippel-Trenaunay-Weber or Sturge-Weber syndromes. The triad of Klippel-Trenaunay-Weber syndrome symptoms includes vascular tumors of the limbs, trunk, or perineum; varicose veins; and bony and/or soft tissue hypertrophy of the extremities. The presenting symptom in patients with Sturge-Weber syndrome is often a facial lesion, most commonly a nevus flammeus located in the V1-V2 dermatomes. Other findings include a history of epileptic seizures, hemiplegia, visual field defects, and glaucoma

Most lymphangiomas are clinically apparent at birth, and almost all are apparent by age 2 years. Most appear as soft doughy masses that are located in the head and neck region, and most have no associated symptoms.

Clinical manifestations are dependent on the flow of lymph within the channels of the lesion. Lymphangiomas may manifest as lymphedema, and larger lesions can involve the skeletal system and cause gross disfigurement. Large malformations in the neck or mediastinum can compromise the airway, leading to stridor, dysphonia, or dyspnea. Lymphangiomas have also been found in patients with Turner syndrome, Klinefelter syndrome, and Noonan syndrome

Treatment

I. Medical Therapy

Vascular tumors

Approximately 75% of hemangiomas involute without intervention and are in anatomically benign locations, making them a cosmetic issue. Regular follow-up visits should be scheduled to monitor the course of the lesion and to provide continuous reassurance to the parents.

Medical intervention is indicated if hemangiomas are located in areas that hinder normal development, interfere with important life processes, or cause severe pain. Such locations include the eye, nose, and airways. Current practices include the following:

- Systemic glucocorticoids are the first-line therapy for lesions that are life threatening or cause severe deformity. Efficacy rates have been reported at 84% with prednisone equivalent doses of 2.9 mg/kg if administered during the proliferative phase [15].
- Triamcinolone is indicated for smaller hemangiomas; intralesional injections administered every 4-6 weeks have been shown to result in faster rates of involution in 30% of patients.
- Interferon alfa-2a is used for life-threatening or deforming lesions that do not respond to glucocorticoid therapy. It is subcutaneously administered at a dose of 1-3 million U/m^2 of body surface area [16].
- Flashlamp-pumped PDL is indicated for superficial residual lesions that remain after involution; this is not effective on deep hemangiomas. Recent studies show no advantage to early laser intervention versus conservative treatment.

Lymphatic malformations

For localized lymphatic malformations, various pharmacologic agents have been used around the world to treat lymphangioma. Some of the agents used are sclerotherapy using boiling water, tetracycline, bleomycin, and cyclophosphamide. Sclerotherapy may also be of value in venous

malformations, acquired digital arteriovenous malformations, and hemangiomas [17].

II. Surgical Therapy

Surgical excision of vascular tumors (hemangiomas) is controversial because most lesions involute with little intervention (75%).

Surgery is indicated if the procedure is anticipated to leave a scar that is more cosmetically acceptable than a scar due to medical therapy or following involution. Furthermore, in the following few specific situations, surgical intervention is considered inevitable if :

(1) An abnormal scar or excess tissue is present after natural involution;

(2) Lesions are ulcerated and bleed excessively or are associated with pain;

(3) Lesions interfere with the development or activities necessary for life, such as lesions of the eye, ear, or larynx.

Lymphatic malformations

Surgical excision for localized lymphangioma if anatomically possible, with total removal of the tumor, leaving behind no cystic epithelium, has been the most reliable procedure. In one study, total

resection led to no recurrences following the procedure. This is compared with recurrences in 6 of 7 patients if excision of only protruding cystic epithelium was performed.

SCLEROTHERAPY

MECHANISM OF ACTION :

Sclerotherapy is the procedure in which chemical energy is used to eradicate a vessel. The chemical energy delivered intravascularly is capable of denaturating biological molecules that comprise the vein wall. For permanent eradication of the vein, it is necessary to produce full mural denaturation or atleast enough mural injury requisite to preclude mural reconstitution. If this is attained, the mural layers will be resorbed, and the lumen that is surrounded will be obliterated. The vein will permanently disappear. The persistence of a vein after treatment is evidence that full mural denaturation was not achieved.

Sclerotherapy targets the mural constituents and not the blood in the vein lumen. Although is unnecessary and undesirable, thombosis almost always develops within the lumen of every treated vein. This is because, once infusion of the sclerosant is terminated, some blood returns into the lumen, which at that time is still intact even though full mural denaturation may have been attained. The presence of sub endothelial collagen and other

coagulation cascade initiators causes the blood to coagulate (i.e., thrombose). Thrombosis does not affect the extent of mural denaturation and does not usually affect the ultimate outcome of treatment [56]. However it prolongs the course of vein resorption and thus the duration for its eventual disappearance.

To limit endothelial injury to a controlled area, we exploit differences in flow dynamics between the abnormal veins being perfused with sclerosant and the adjacent normal vessels that should not be sclerosed. A thorough understanding of the mechanism of action of the principal sclerosing agents is essential, as is a firm grasp of the biophysical principles underlying the techniques of sclerotherapy.

Volume dilution and patient positioning[18]

Sclerosant is diluted with blood as it diffuses away from the site of injection, thus if a strong sclerosant is injected there will be three zones of action. In zone 1, vascular endothelium is irreversibly injured: the vessel will be fully sclerosed and eventually will be completely replaced by a fibrous tissue. In zone 2, vascular endothelium is injured, and the vessel will be partially or completely thrombosed but will eventually recanalize. In zone 3

the sclerosant will be diluted below its injurious concentration, and there will be no endothelial injury.

Dilution by diffusion from the injection site[18]

Because dilution of the sclerosant with blood occurs immediately upon injection, the original injected concentration is of no real importance. What is important is the diluted concentration of sclerosant at the surface of the endothelium. An injected concentration that is perfectly effective in a spider vein (where sclerosant displaces blood rather than mixing with it) may be ineffective in a reticular feeding vein or a truncal varix simply because dilution reduces the final concentration so low that there will be no endothelial injury whatsoever (no zone I or zone II). If the injected concentration is too high, dilution will leave the final concentration so high that endothelial damage will occur where it is not wanted (zone I and zone II are too large). If the injected concentration is just right, dilution will leave a final concentration that is sufficient to injure the local varicose endothelium, but not high enough to damage normal superficial or deep veins (most of the varicose vessel falls into zone I, a small amount falls into zone II, and all normal vessels fall into zone III).

When we select a particular volume and concentration of a chemical agent with which to sclerose a vessel, we are explicitly or implicitly adjusting the injected concentration and volume to take into account the dilution that will occur when the sclerosant is mixed with blood immediately after injection. We also must take into account the further dilution that will occur as the sclerosant flows or diffuses away from the site of injection. The importance of patient positioning in determining dilutional volume often is not properly appreciated by the novice in phlebology.

Because of the cylindrical geometry of blood vessels, the volume contained in a vessel depends on the square of the vessel radius: the volume of any cylinder is calculated as **pi * r2 * L** (where r is the radius and L is the length of the vessel). Vessels collapse to a smaller radius when the legs are elevated, thus the volume contained is reduced dramatically. For this reason, the position of the patient has a very powerful effect on the final diluted concentration of sclerosant at the surface of the vessel endothelium.

EFFECT OF POSITION ON VARICOSE GEOMETRY[18]

- **Standing**

For a standing patient with a superficial varicosity of 2 cm in diameter, the final concentration at a distance from the injection site of 10 cm (4 inches) is 30 times lower than the initial concentration. Doubling the initial concentration serves only to double the final concentration, which will still be 15 times weaker than the concentration in the syringe. In other words, if 1 cc of a 3% solution is injected, the final concentration at the endothelial surface is 1% at a distance of 1 cm from the injection point, 0.5% at a distance of 2cm, 0.25% at a distance of 4 cm, and 0.2% at a distance of 5cm (2 inches) from the injection point. As we shall see, this means that it is very difficult to achieve sclerosis of a large vessel by injecting detergent sclerosants with the patient in a standing position: if the highest available concentration is injected, the dilution factor may still drop the final concentration below the threshold of effectiveness within 1.5 inches from the injection site.

- **Supine**

Varicose vessels that bulge when the patient is standing may collapse when the patient is supine, but duplex ultrasound readily demonstrates that the veins are not empty of blood. Both varicose and normal vessels contain a significant volume of blood with the legs extended in the supine position. A bulging varicosity that has a diameter of 2 cm in the standing position may have a diameter of 1 cm in the supine position and of 0.5 cm or less when the legs are elevated as high as possible. With such a patient in the supine position, injection of 1 cc of a 3% solution leads to a final concentration of approximately 1.7% at a distance of 1 cm and a concentration of about 0.6% at a distance of 5 cm (2 inches). This supine technique limits dilution enough to allow successful sclerosis of large vessels using detergent solutions, so long as sufficient concentrations and volumes of sclerosants are injected. The only problem is that if an injection of sclerosant at a high initial concentration is made directly into a perforating vessel, so that sclerosant flows directly into the deep system, dilution within the deep vessel will still permit zone I and zone II endothelial injury for a short distance within the deep vein. This can lead to deep vein valve damage and chronic venous insufficiency, to deep vein thrombosis, and to life-threatening pulmonary embolism.

- **Legs elevated**

In contrast to the standing and supine positions, when a patient lies supine and the legs are raised vertically so that they are well above the central circulation, most superficial varices collapse to the point where they no longer contain any significant volume of blood. Repeating the calculation above for a patient in this position, injection of 1cc of a 3% solution leads to a final concentration of 2.5% at a distance of 1 cm from the injection, and a final concentration of 1.6% at a distance of 5 cm (2 inches). In fact, the final concentration will still be above 1% at a distance of 10cm from the injection site. Because the superficial varicosity is collapsed, there is very little dilution with distance so long as the sclerosant stays within the floppy-walled varicosity.

Although flow measurements reveal little or no spontaneous flow through varices and smaller superficial veins when the patient is in the leg-up position, a substantial intravenous volume and a substantial rate of flow still persists in the deep veins and in normal larger superficial veins, which have less collapsible walls. This difference between the volumes contained in floppy superficial varicose veins and the volumes contained in normal surface veins and deep veins may be exploited to cause damage that is

almost perfectly localized to superficial varices. If an elevated, empty varicose vessel is perfused with a concentration of sclerosant so low that even without dilution it is just barely sufficient to cause endothelial injury, then any further dilution will reduce the concentration below the threshold of injury. Because larger superficial vessels and deep vessels continue to carry a volume of blood in the leg-up position, any sclerosant passing into these vessels will immediately be diluted to a safe and noninjurious concentration, sparing the endothelium of vessels that we wish to preserve. Injection of this 'threshold' concentration directly into a perforating vein (or even directly into a deep vein) will not cause any deep vein injury.

The perfect sclerosant

The best imaginable sclerosant would have no systemic toxicity. It would be effective only above some threshold concentration, so that its effects could be precisely localized through dilution. It would require a long period of contact to be effective, so that it would be relatively more effective in areas of stasis and relatively safer in the deep veins where there is high flow. It would be non-allergenic. It would be strong enough to sclerose even the largest vessels, yet it would produce no local tissue injury if extravasated. It would not cause staining or scarring. It would not cause telangiectatic matting. It would be perfectly soluble in normal saline. It

would be painless upon injection. It would be inexpensive. It would be approved by the United States Food and Drug Administration.

No currently available sclerosant possesses all of the attributes of the perfect sclerosing agent. All currently available sclerosants fall short in one way or another, yet the variety of available agents is such that virtually every situation in which sclerotherapy is indicated can be safely and effectively handled by one or another of the available sclerosants, used alone or in combination.

TYPES OF SCLEROSANTS :

(I) DETERGENTS

These are fatty acids and fatty alcohols. Detergent sclerosants work by a mechanism known as protein theft denaturation, in which an aggregation of detergent molecules forms a lipid bilayer in the form of a sheet, a cylinder, or a micelle, which then disrupts the cell surface membrane and may steal away essential proteins from the cell membrane surface. Unlike many other agents, the detergent sclerosants do not cause hemolysis, nor do they provoke direct intravascular coagulation.

Determinants of activity of detergent solutions [18]

(a) Concentration

At low concentrations, most detergent molecules are individually dissolved in solution, and there are very few micellar aggregates. When the concentration reaches some threshold (known as the critical micellar concentration, or CMC) nearly all further detergent molecules added to the solution will enter into micelles. Micelles can cause protein theft denaturation, but individual detergent molecules have no toxicity to the vascular endothelium, thus for each detergent sclerosant, there is some threshold concentration below which the agent causes no injury. This physical property means that detergent sclerosants offer significant benefits over most of the agents previously used, because they are potent agents that nonetheless have a clear-cut threshold below which they have absolutely no injurious effect on venous endothelium.

(b) Temperature

The solubility of detergents is inversely temperature dependent. Because of the highly polar nature of water, and the entropic dependence of the hydrophobic effect, detergent molecules are much more soluble in cold

solutions than in hot ones. This effect is easily seen in everyday life: dishwashing detergent produces a large amount of persistent foam in warm water, while cold water rinses away the soapy foam easily. The solubility of sclerosing agents such as polidocanol is likewise much higher in cold solutions, and because single dissolved molecules are ineffective, the strength of the sclerosing effect is higher at warmer temperatures.

(c) Mixing

Detergent micellar formation can reach a maximum level based upon the temperature and upon the concentration of the detergent in solution. Micellar formation is a steric process, however, and the geometry of macro assemblages often prevents maximal micellar formation. The surface area of lipid bilayer structures such as sheets, cylinders, and micelles is maximized when the solution is shaken to produce foam. Because it is the surface of these structures that causes protein theft denaturation, a solution that has been shaken will be a more effective sclerosant than one that has not. Unfortunately, foamy bubbles that are injected into spider veins or varicose veins can pass through a patent foramen ovale to lodge in the ocular and cerebral circulation, where they have produced temporary ischemic attacks with temporary blindness and other central nervous system effects.

Currently available detergent agents

1) Sodium morrhuate

2) Ethanolamine oleate

3) Sodium tetradecyl sulfate (Sodium 1-isobutyl-4-ethyloctyl sulfate) a synthetic long chain fatty acid that has seen extensive industrial use as a synthetic surfactant (soap). It is sold for medical use as a solution of up to 3% concentration with 2% benzoyl alcohol used as a stabilant. It is effective as a venous sclerosing agent in concentrations from 0.1% to 3%. The principal clinical problems with the drug are a tendency to cause hyperpigmentation in up to 30% of patients, a significant incidence of epidermal necrosis upon extravasation, and occasional cases of anaphylaxis.

4) Polidocanol (hydroxy-polyethoxy-dodecane) is a synthetic long-chain fatty alcohol

(II) HYPERTONIC AND IONIC SOLUTIONS

Strong solutions of hypertonic saline and other salt solutions are part of a class of solutions that are often referred to as osmotic sclerosants. These solutions have long been regarded as causing endothelial death by osmotic cellular dehydration. Although it is true that osmotic dehydration at the point

of injection is sufficient to rupture red blood cells and to dehydrate some nearby endothelial cells, the evidence suggests that these sclerosants are effective even after dilution has reduced the osmotic gradient far too low to account for the effects seen. Thermodynamic and physical chemical calculations suggest that these and other strong ionic solutions probably work by causing conformational denaturation of cell membrane proteins in situ. Like the detergents, they can be diluted to the point where they have no further cellular toxicity.

1) Hypertonic saline

2) Sclerodex

Sclerodex is a mixture of 25% dextrose and 10% sodium chloride, with a small quantity of phenethyl alcohol.

3) Polyiodinated iodine

(III) CELLULAR TOXINS

Other chemical sclerosants exist that probably act by a direct or indirect chemical toxicity to endothelial cells: by poisoning some aspect of cellular activity that is necessary for endothelial cell survival. Such agents are less useful to the extent that they also poison other bodily cells. They

also lack another of the key attributes of a good sclerosant: they remain toxic to some degree even after extreme dilution, so that there is no real threshold below which injury will not occur.

FOAM SCLEROTHERAPY

In foam sclerotherapy, the sclerosant is mixed with air or physiological gases. Foamed sclerosant blocks flow of blood into and out of the treated site and hence avoids dilution of the drug. Foam sclerotherapy is more efficacious than the direct injection of the sclerosants [3]. Air in the foam prolongs the contact of the agent with the endothelium. Maximum sclerosant action can be obtained at lesser concentration and quantity. Foamed agent exerts its effect at microcirculation, which is inaccessible to other procedures.

INDICATIONS FOR SCLEROTHERAPY :

- Varicose veins
- Telangiestasias
- Hemangiomas
- Lymphangiomas
- Pyogenic granulomas

Veins of any size from small venous telangiectasias to large varicose veins and at any size from the face and scalp to the foot and ankle can be permanently eradicated by sclerotherapy. In case of incompetent venous junctions or incompetent perorating veins communicating with the varicose vein, interruption of these points of reflux (e.g. surgical ligation) and division should be considered before sclerotherapy. Hence sclerotherapy is effective in eradicating a segment of an axial vein, but unlike ligation and division, it cannot reliably target a finite point (e.g. saphenofemoral or saphenopopliteal junction, or a short segment of a perforating vein) without also infusing sclerosant into the deep venous system. In addition to the cosmetic purposes sclerotherapy serves, it is also performed to treat the soreness, aching, muscle fatigue, and leg cramps that often accompany small- or middle-sized varicose veins in the legs.

CONTRAINDICATIONS FOR SCLEROTHERAPY :

- Allergy to the sclerosants
- Pregnancy
- Lactation
- Deep vein thrombosis
- Congenital and acquired coagulation abnormalities

MATERIALS AND METHODS

MATERIALS AND METHODS

Study type : Prospective non-randomized interventional study

Study population : 40 patients of both sex; age group : 15 to 65 years

{ 28 with varicose veins with complications

10 with hemangioma

2 with lymphangioma }

Study period : January 2009 to September 2010 (18 months)

First 6 months : Interventional period

Next 12 months : follow-up period

Place of study : Department of Dermatology,

Govt. Stanley Medical College, Chennai.

The patients chosen for the study, were from the general pool attending the Dermatology out patient department. Initially, 70 patients with varicose veins who came with complaints of pain, itching, edema, discolouration, dilated veins over legs; 15 with hemangioma presenting with

swelling and 2 with lymphangioma were subjected to the pre-sclerotherapy consultation, assessment and investigations as given below. Out of them, 28 patients with varicose veins (legs), 10 patients with hemangioma (tongue, gluteal region, hand, trunk of size varying from a minimum of 3 x 2cm to a maximum of 12x8cm) and 2 with lymphangioma (posterior one third of tongue and gluteal region), who were enrolled for the study, according to the inclusion and exclusion criteria. The mean age group was 32.7 years (15 – 65 years) of both sexes.

PRE – SCLEROTHERAPY CONSULTATION :

- Thorough explanation of the procedure, course of treatment and potential adverse effects were explained
- Informed and written consent was obtained
- A thorough history regarding the onset, duration, etiological factors, complications, other systemic illnesses, hypersensitivity to sclerosants, pregnancy, lactation and past history regarding any modes of treatment for the condition was taken (ANNEXURE - I)

PRE – SCLEROTHERAPY ASSESSMENT :

A meticulous clinical examination as given in ANNEXURE – I was done with the following tests for varicose veins :

- Tourniquet test
- Brodie-Trendelenberg test
- Fegan's test
- Pratt's test
- Perthes test
- Homan's sign
- Moses sign
- Schwartz's test

Investigations :

In all patients with varicose veins, Doppler ultra sonogram of venous and arterial systems of both lower limbs, ultra sonogram of abdomen and pelvis and coagulation profile was done. In patients with hemangiomas and lymphangiomas, contrast enhanced CT scan, MRI and coagulation profiles were done (as required).

With the history, clinical examination and the aid of laboratory investigations, patients were selected according to the inclusion and exclusion criteria.

Inclusion criteria :

1. Willing to be enrolled for the complete study period
2. superficial varicosities with no junctional refluxes
3. reticular veins
4. patients unfit for surgical procedure
5. recurrence after surgery
6. post surgical ligation of major refluxes

Exclusion criteria :

1. deep venous thrombosis
2. veins with reflux from the greater or lesser saphenous junction per se
3. perforator incompetence
4. coagulation abnormalities
5. allergy / hypersensitivity to the drug
6. pregnancy

7. lactation
8. on oral contraceptive pill or hormone replacement therapy

PROCEDURE :

Requirements (Figure 1) :

- Sclerosing agent – sodium tetradecyl sulphate (3%) EDA and FDA approved detergent sclerosant
- 0.9% saline as diluent
- 5 ml syringes
- Scalp vein set
- Sterile bowl for sclerosant
- Gloves
- Alcohol pads
- Gauze
- Elasto-crepe bandage roll

Figure 1 - Sclerotherapy supply tray

FOAM SCLEROTHERAPY TECHNIQUE :

➢ Positioning of patient :

Patient should be recumbent [18] during the procedure, for comfort and to promote venous emptying.

➢ Visualization of the vein :

The skin where the injection is to be given is wiped with surgical spirit to remove oil, dirt and for easy visualization of the vein (as wetting of the skin lowers its refractive index)

➢ Preparation of the foam sclerosant :

1. Sodium tetradecyl sulphate 3% (STS), which is an EDA and FDA [19] approved detergent sclerosant is diluted with normal saline to get 0.5 – 2% solution.
2. Quantity per session and concentration used were based on the diameter of the vessel, size of the hemangioma and the response to treatment. Quantity used by us per session was 1 – 3 ml.
3. Concentration of the sclerosant :

   The concentration of injected sclerosant is determined mainly

by the mural thickness of the vein, which is directly correlated with the lumen diameter as given in table I. The greater the vein diameter, the greater the concentration of sclerosant that will be needed. Injection of too low a concentration will be inadequate to produce full mural denaturation. Injection of too high a concentration can produce extra vascular tissue devitalisation.

TABLE - I

Diameter of the vessel	Concentration of sclerosant
< 0.5mm	0.1 %
0.6 – 1.5mm	0.25 %
1.5 - 4mm	0.5 – 1 %
4 -6 mm	1 – 2 %

Calculation of dilution volumes for sclerosants is done using the formula :

$$\mathbf{C_f = C_i [V_s / (V_s + V_d)]}$$

C_f – final concentration (in %) of the sclerosant

C_i - initial concentration (in %) of the sclerosant

V_s – initial volume (in ml) of undiluted sclerosant

V_d – diluent volume (in ml) of 0.9% saline solution

For example, to prepare 0.5% solution of STS, using 1 ml of 3% STS, the volume of 0.9% normal saline to be added is calculated using the above formula :

$C_f = C_i [V_s / (V_s + V_d)]$

$0.5 = [3] [1 / (1 + V_d)]$

$0.5 / 3 = 1 / (1 + V_d)$

$0.5 + 0.5 V_d = 3$

$V_d = 5$ ml

Hence 5 ml of 0.9% normal saline is needed to make 3% STS (1 ml) to 0.5%

➢ Foaming of the sclerosant :

Foaming is done by **'Tessari' method** [20], in which a 5 ml syringe is filled with room air, another syringe is filled with 1 ml of the diluted sclerosant and the two syringes are connected to a 3 way stop cock (Figure 2). Foaming is done by to and fro movement of both the syringes (Figure 3).

➢ Injection of the sclerosant :

1. The cannula of the needle is introduced into the vein such that it enters parallel to the vessel's course. The bevelled edge is positioned to face upwards (towards the skin surface), so that even if extravasation occurs, it will be superficial and hence easily visualized as a bleb, and not deposited deeper.
2. In case of varicose veins, cannulation is done from the proximal end (Figure 4) and hemangiomas are cannulated at the base [21] (Figure 5).

Foaming of the sclerosant done by ‘Tessari’ method

Figure 2

Figure 3

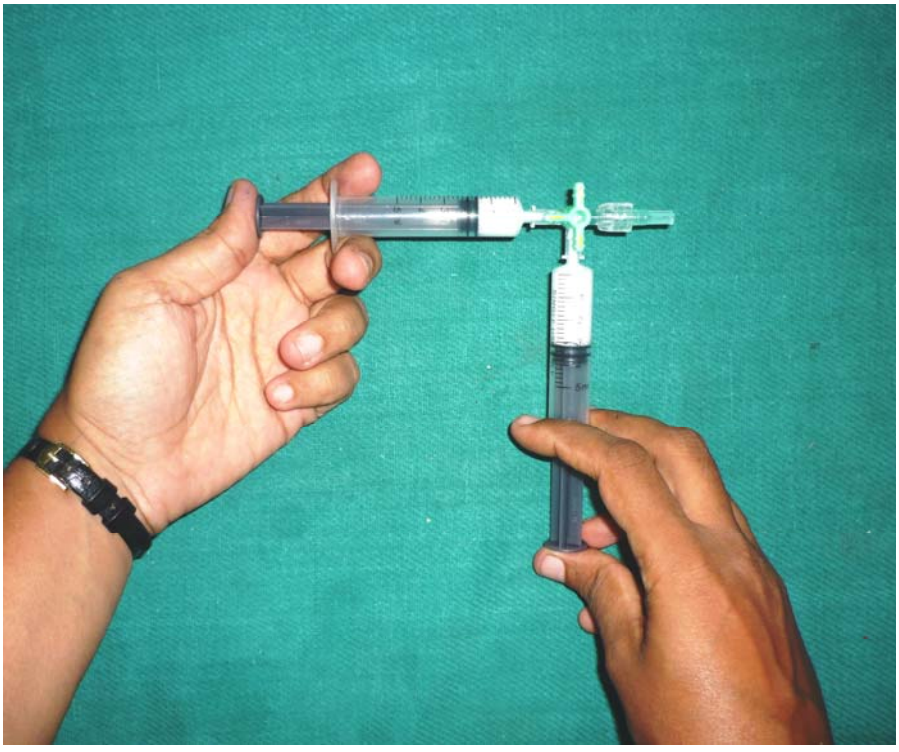
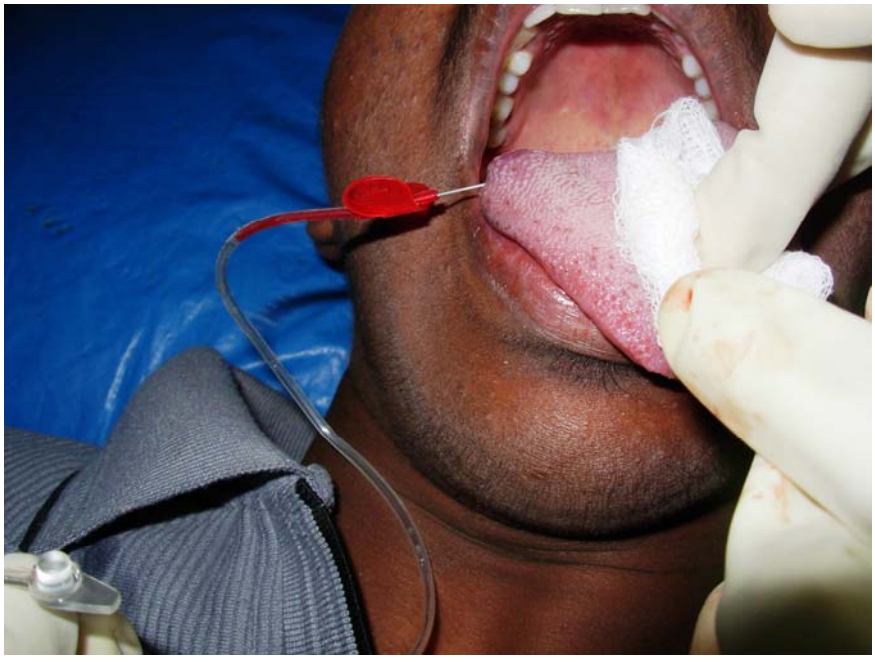


Figure 4 – cannulation of varicose vein

Figure 5 – cannulation of hemangioma



3. The vein is pierced and after confirming the intraluminal position of the cannula, the limb is elevated to 45^{o} [18] to further empty the vein and the diluted sclerosant is slowly introduced into the vein.
4. Once extravasation or high injection pressure is experienced, further injection is stopped.
5. Immediately after the procedure, a circumferential compression bandaging [22] is done using elasto-crepe bandage (Figure 6).

POST PROCEDURE ADVISES:

- To wear compression stockings at all times, day and night, at least for 2 weeks.
- To keep their legs elevated
- To carry out normal daily activities
- To avoid strenuous exercise
- To avoid standing still for long periods
- Bed rest was not recommended.

Figure 6 – Post sclerotherapy compression bandaging

FOLLOW UP :

Follow up was done at every 2 weeks interval assessing the fibrosis of the vessels, reduction in size of the hemangioma and local or systemic complications (if any). Procedure was repeated at an interval of 2 weeks depending upon the response which was judged clinically. Doppler ultra sonogram was repeated at the end of 3^{rd}, 6^{th}, 12^{th},18^{th} months.

RESULTS

RESULTS

Total no. of patients in the study group :

Male : 30

Female : 10

Out of 28 patients with varicose veins –3 patients presented with pain over the dilated veins, 3 with swelling of feet, 2 with itching, 4 with discolouration of legs, 4 with leg ulcer, 12 with stasis dermatitis.

TABLE 1 – SYMPTOM-WISE DISTRIBUTION OF PATIENTS

SYMPTOM	NO. OF PATIENTS
Pain	3
Swelling of feet	3
Itching	2
Discolouration	4
Leg ulcer	4
Stasis dermatitis	12

TABLE 2 - VESSEL CLASSIFICATION [57]

VESSEL TYPE	DEFINITION	VESSEL DIAMETER (mm)	NO.OF PATIENTS
1	Spider veins	0.1 – 1.0	-
1-A	Telangiectatic matting	<0.2	1
1-B	Telangiectasia communicating with saphenous system	0.2 – 1.0	1
2	Mixed telangiectatic/varicose veins	1.0 – 6.0	4
3	Non-saphenous varicosities (reticular veins)	2.0 – 8.0	3
4	Saphenous varicose veins	>8.0	19

TABLE 1 – SYMPTOM-WISE DISTRIBURION OF PATIENTS

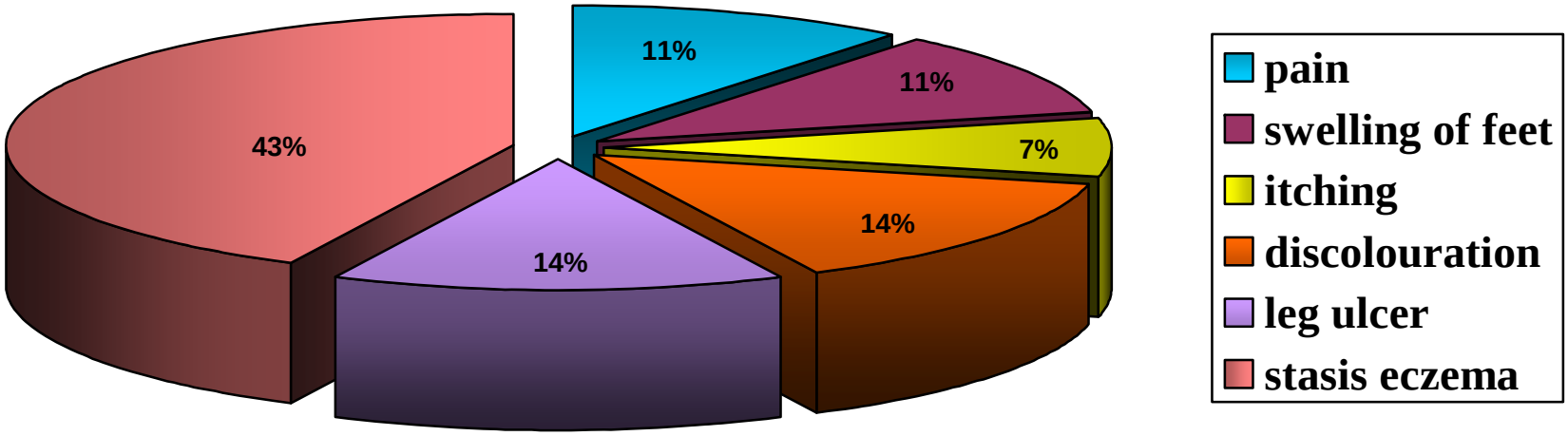
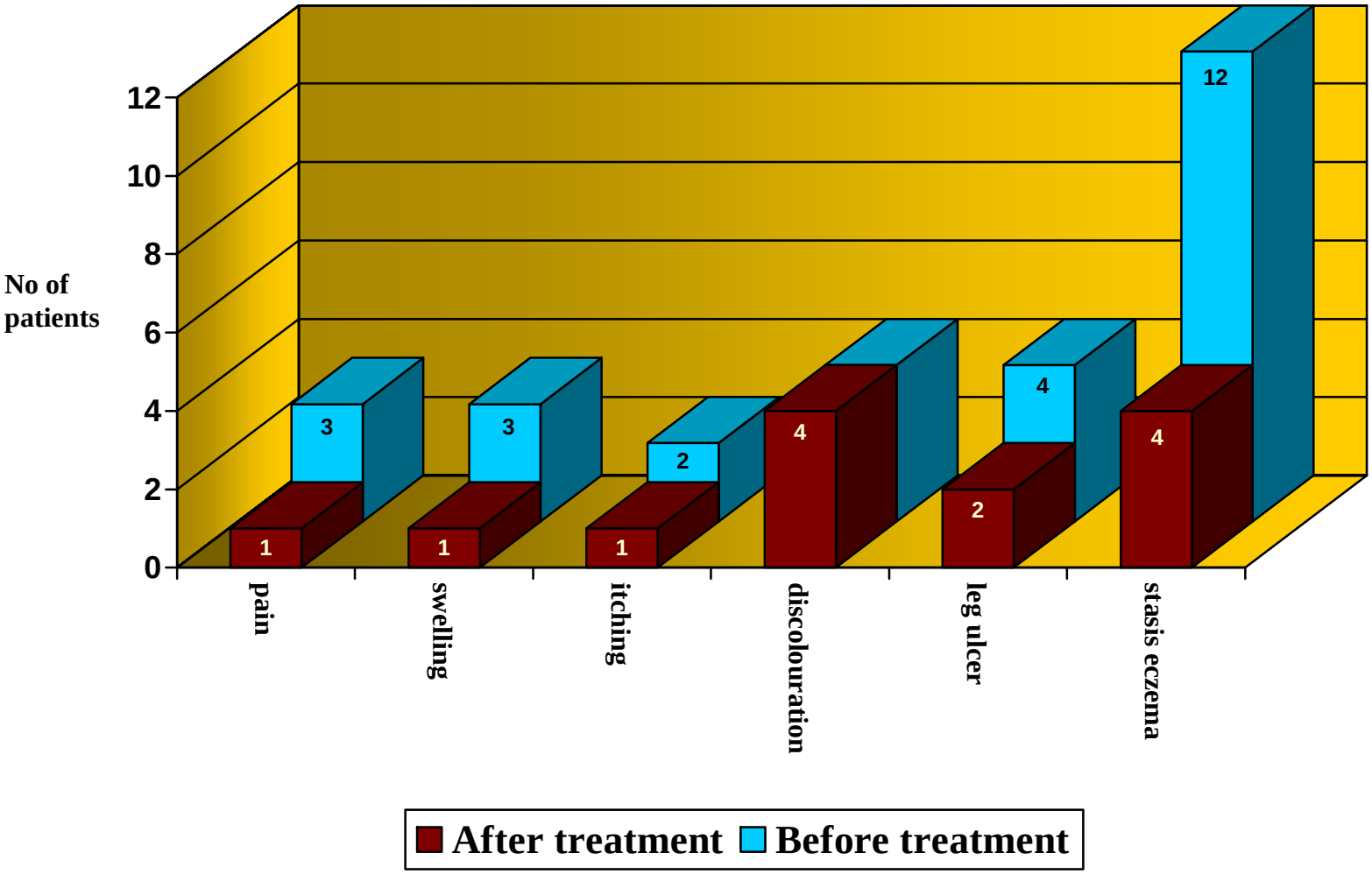


TABLE 3 - PERCENTAGE RESPONSE OF SYMPTOMS AFTER TREATMENT

SYMPTOM	NO. OF PATIENTS WITH THE SYMPTOM BEFORE TREATMENT	NO. OF PATIENTS WITH THE SYMPTOM AFTER TREATMENT	PERCENTAGE IMPROVED (%)
Pain	3	1	66.7
Swelling	3	1	66.7
Itching	2	1	50
Discolouration	4	4	0
Leg ulcer	4	2	50
Stasis eczema	12	8	66.7

CHART (TABLE 3) - RESPONSE OF SYMPTOMS AFTER TREATMENT :



At the end of 3 months (completion of 2 sittings):

4 patients with varicose veins, 2 patients with hemangioma showed complete resolution; near complete resolution was seen in 16 cases with varicose veins, 6 with hemangioma and 1 with lymphangioma; partial resolution was seen in 8 with varicose veins, 2 with hemangioma and 1 with lymphangioma.

At the end of 6 months (completion of 5 sittings):

23 patients with varicose veins, 6 with hemangioma and 1 with lymphangioma showed complete resolution; near complete resolution was seen in 3 cases with varicose veins, 4 with hemangioma and 1 patient with lymphangioma; partial resolution was seen in 2 patients with varicose veins.

At the end of 12th month :

27 patients with varicose veins, 7 with hemangioma and 1 with lymphangioma showed complete resolution; near complete resolution was seen in 1 patient with varicose vein, 3 patients with hemangioma and 1 patient with lymphangioma.

At the end of 18th month :

Out of 28 patients with varicose veins who showed complete resolution, 3 had recurrence, 7 with hemangioma and 1 with lymphangioma showed complete resolution; near complete resolution was seen in 3 patients with hemangioma and 1 patient with lymphangioma. (Table 4)

CRITERIA FOR RESOLUTION :

COMPLETE RESOLUTION :

- varicose veins – complete obliteration of vein with no flow
- hemangioma and lymphangioma – complete absence of flow in the vessels (Doppler)

NEAR COMPLETE RESOLUTION :

- varicose veins – decrease in luminal diameter more than 75% of initial diameter (by Doppler)
- hemangioma and lymphangioma - decrease in size more than 75% of initial (clinical) and/ or decrease in flow more than 75% of initial (Doppler)

PARTIAL RESOLUTION :

- varicose veins – decrease in luminal diameter less than 75% of initial (Doppler)
- hemangioma and lymphangioma - decrease in size less than 75% of initial (clinical) and/ or decrease in flow less than 75% of initial (Doppler)

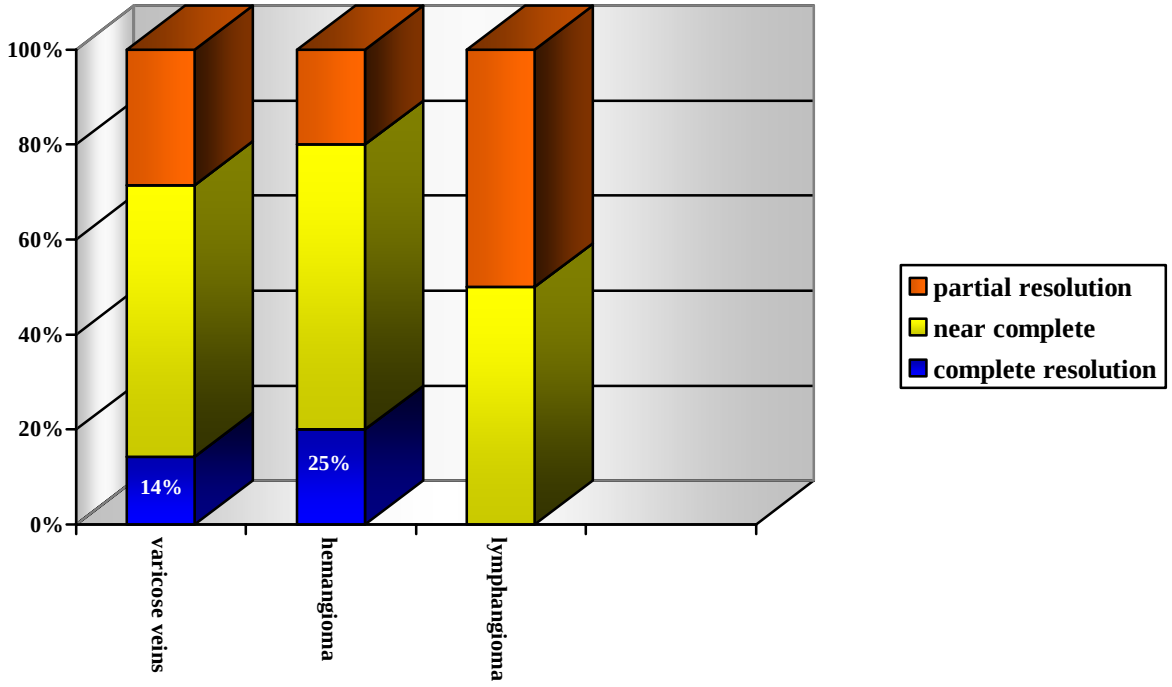
RECURRENCE :

Varicose veins, hemangioma & lymphangioma } - Detection of flow in vessels which had attained complete resolution

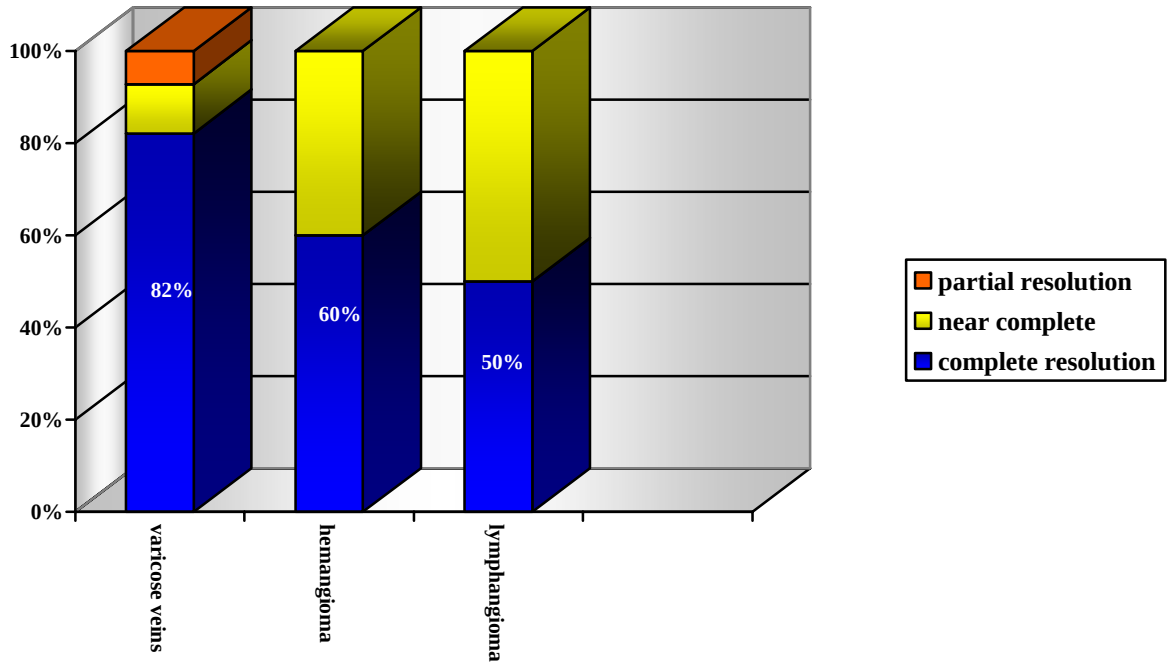
RESPONSE	VARICOSE VEINS (months)				HEMANGIOMA (months)				LYMPHANGIOMA (months)			
	3	6	12	18	3	6	12	18	3	6	12	18
Complete	4	23	27	25	2	6	7	7	-	1	1	1
Near complete	16	3	1	-	6	4	3	3	1	1	1	1
Partial	8	2	-	-	2	-	-	-	1	-	-	-
Recurrence	-	-	-	3	-	-	-	-	-	-	-	-

Table 4 : Response to treatment during follow-up

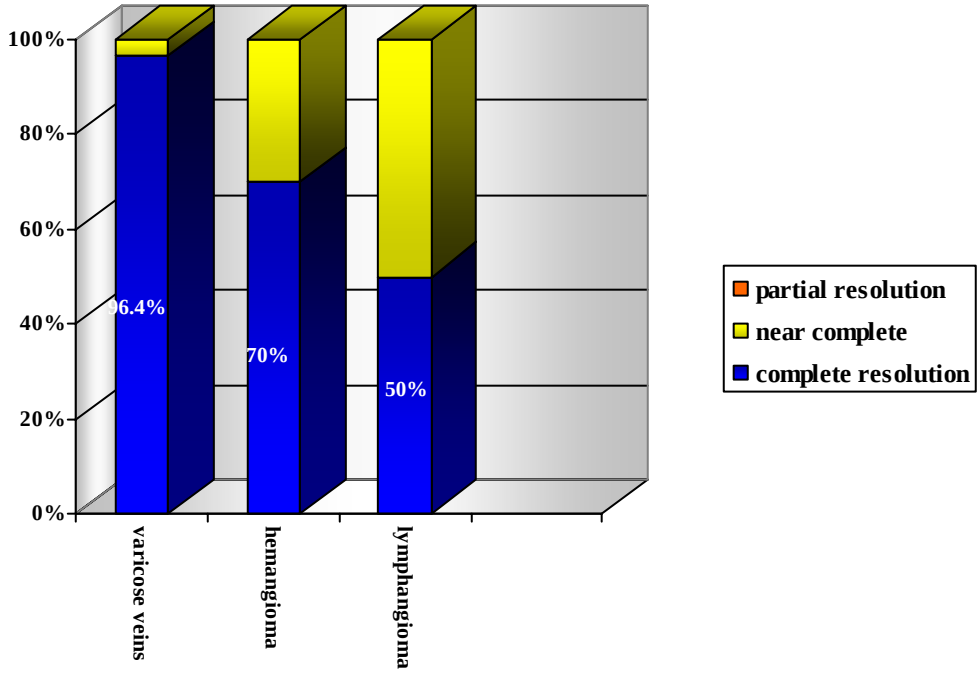
At the end of 3 months (completion of 2 sittings) :



At the end of 6 months (completion of 5 sittings):



At the end of 12th month :



At the end of 18th month :

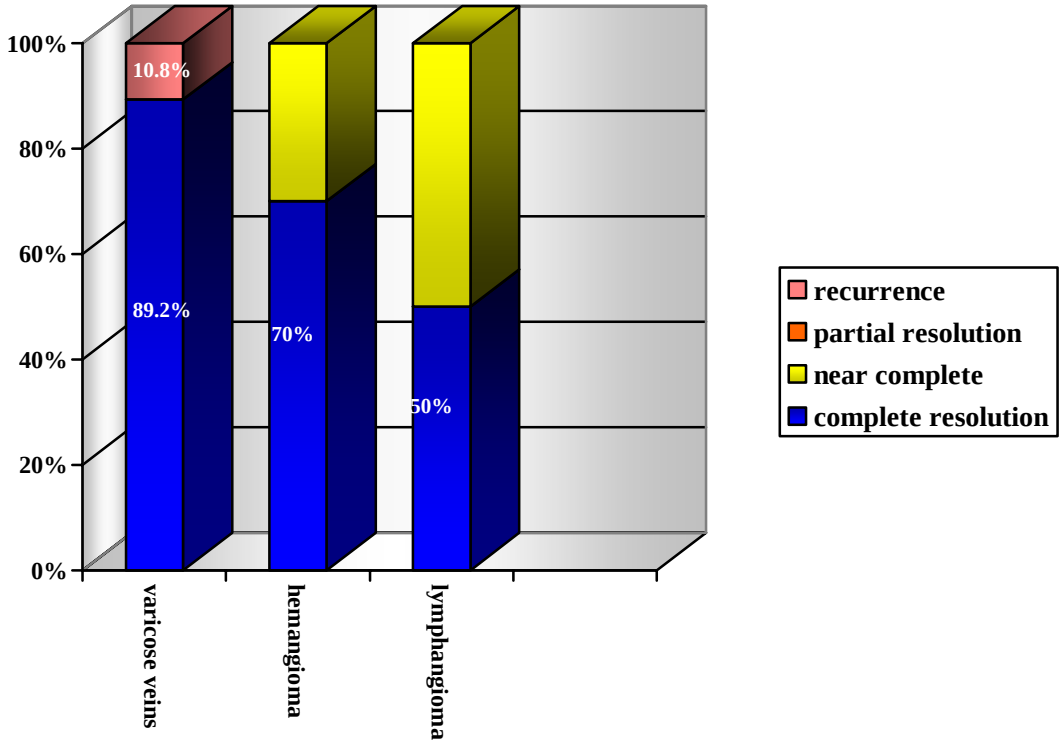


Figure 6-A : Localized varicose vein Rt. Leg –Before treatment

Figure 6-B : After 2 sittings of sclerotherapy

Figure 7-A : Varicosity of great saphenous vein Lt. Leg – Before treatment

Figure 7-B : After 4 sittings of sclerotherapy

Figure 8-A : Localized varicosity of Lt. great saphenous vein

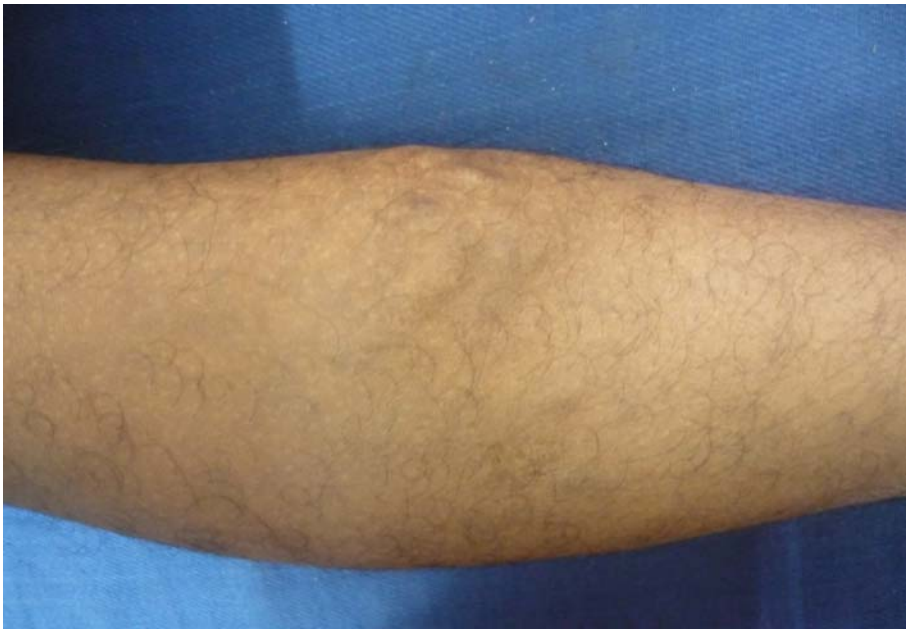


Figure 8-B : After 4 sittings of sclerotherapy

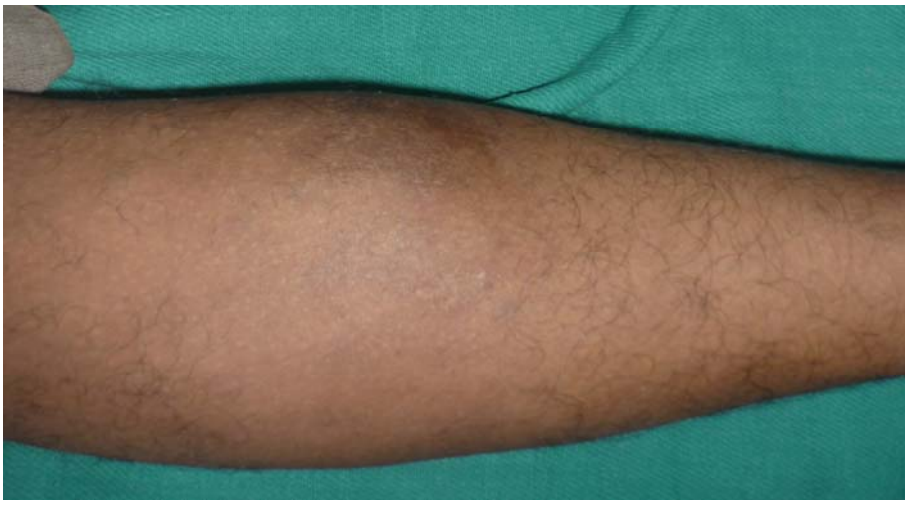


Figure 9-A : Lt. leg varicose vein with stasis eczema and venous ulcer

Figure 9-B : After 5 sittings of sclerotherapy – healing of stasis eczema & ulcer

Figure 10-A : Lt. leg varicose vein - before treatment

Figure 10-B : After 3 sittings of sclerotherapy

Figure10-C : After 5 sittings of sclerotherapy

Figure 11-A : Varicosity of small saphenous vein Lt. leg

Figure 11-B : After 3 sittings of sclerotherapy

Figure 12-A :Localized varicosity of Rt. Great saphenous vein

Figure 12-B : After 5 sittings of sclerotherapy

Figure 13-A : Lymphangioma – posterior 1/3rd of tongue

Figure 13-B : After 5 sittings of sclerotherapy

Figure 14-A : Hemangioma – tongue

Figure14-B : After 6 sittings of sclerotherapy

Figure 15-A : Hemangioma – gluteal region

Figure 15-B : After 6 sittings (partial resolution)

Figure 16-A : Hemangioma Lt. hand

Figure 16-B : After 2 sittings of sclerotherapy

INTERPRETATION OF RESULTS :

The average no. of sittings required for complete resolution :

- 4.2 sittings for varicose veins
- 3.9 sittings for hemangiomas
- 5.5 sittings for lymphangioma

This in turn depends upon the diameter and length of the veins and the size of the hemangioma.

COMPLICATIONS OBSERVED :

- The following complication were noted during the study :

COMPLICATION	NO. OF PATIENTS
Swelling of the treated limb	3
Pain over the treated site	3
Cutaneous ulceration	1
Staining of skin at and around treated site	4

- No immediate complications like hypersensitivity reactions, were noted.
- No serious complications like deep vein thrombosis or pulmonary edema were noted.
- Pain and swelling after treatment persisted for 1-2 weeks and subsided with oral analgesics.
- Post-sclerotherapy staining (Figure 17) gradually faded in 3 patients over a period of 6 – 10 months and was persistent in one patient even at the end of 18 months
- The cutaneous ulceration (Figure 18) which had occurred secondary to extravasation of the sclerosant healed in 2 months with conservative management (Figure 18A).

Figure 17 : Post-sclerotherapy staining of skin

along the course of the vein

Figure 18-A : Cutaneous ulceration – due to extravasation

Figure 18-B : Healing of the ulcer

DISCUSSION

DISCUSSION

Varicose veins and telangiectatic leg veins have plagued both men and woman since humans first assumed an upright posture.

According to Tollins SH [23], varicose veins occur more commonly in women than men with a ratio 4:1 to 5:1, whereas in this study we have found an increased incidence in men 3.7:1 (male : female).

Though there are various treatment options available for varicose veins as discussed above, veins less than 5mm in diameter and those not associated with junctional refluxes or perforator incompetence can be treated by non-surgical methods.

Sclerotherapy is the "gold standard" and is preferred over laser for eliminating large spider veins (telangiectasiae) and smaller varicose leg veins [24]. The foam sclerosant drug is more efficacious than the liquid one in causing sclerosis, for it does not mix with the blood in the vessel and in fact displaces it, thus avoiding dilution of the drug and causing maximal sclerosant action. It is therefore useful for longer and larger veins [25]. Experts in foam sclerotherapy have created “tooth paste” like thick foam for their injections, which has revolutionized the non-surgical treatment of varicose veins [26].

A study by Kanter and Thibault in 1996 reported a 76% success rate at 24 months in treating saphenofemoral junction and great saphenous vein incompetence with STS 3% solution [27].In our study 89.2% success rate was noted. 70% of patients with hemangioma and 50% with lymphangioma showed complete resolution over a period of 18 months.

Padbury and Benveniste [28] found that ultrasound guided sclerotherapy was effective in controlling reflux in the small saphenous vein. Barrett et al found that microfoam ultrasound guided sclerotherapy was "effective in treating all sizes of varicose veins with high patient satisfaction and improvement in quality of life"[25].

A Cochrane Collaboration review of the medical literature concluded that "the evidence supports the current place of sclerotherapy in modern clinical practice, which is usually limited to treatment of recurrent varicose veins following surgery and thread veins."[29] A second Cochrane Collaboration review comparing surgery to sclerotherapy concluded that sclerotherapy has greater benefits than surgery in the short term but surgery has greater benefits in the longer term. Sclerotherapy was better than surgery in terms of treatment success, complication rate and cost at one year, but

surgery was better after five years. However, the evidence was not of very good quality and more research is needed.[30]

The work of Cabrera [31] and Monfreaux in utilizing foam sclerotherapy along with Tessari's "3-way tap method" [20] of foam production further revolutionized the treatment of larger varicose veins with sclerotherapy

George Fegan in the 1960s reported treating over 13,000 patients with sclerotherapy, significantly advancing the technique by focusing on fibrosis of the vein rather than thrombosis, concentrating on controlling significant points of reflux, and emphasizing the importance of compression of the treated leg [32].

The degree of compression necessary after sclerotherapy directly correlates with the maximal intravascular pressure that the vein sustains. External compression must exceed the maximal intravascular pressure to prevent any blood return into the lumen of denatured vein. A class 2 gradient compression hosiery is advocated in many studies with a pressure of 30 to 40mm Hg.

Compression serves atleast five main purposes:

- If adequate, results in direct apposition of the treated vein walls to produce a more effective fibrosis [33].

- Decreases the extent of thrombus formation [34].
- Decreases the incidence of post-sclerotherapy pigmentation [35].
- Prevents telangiectatic matting [36].
- Improves function of calf muscle pump [37].

ADVERSE SEQUELAE & COMPLICATIONS OF SCLEROTHERAPY

[53], [54], [55]

- Temporary hyperpigmentation
- Temporary swelling
- Telangiectatic matting
- Pain
- Localized urticaria
- Tape / compression blister
- Tape compression folliculitis
- Cutaneous necrosis
- Systemic allergic reaction
- Thrombophlebitis
- Arterial injection
- Pulmonary embolus
- Stroke

HYPERSENSITIVITY REACTION :

Immediate hypersensitivity to sclerosing agent or its preservative could appear as a rare phenomenon[38], which can be treated with subcutaneous epinephrine (0.5ml of epinephrine 1:1000)

VASOVAGAL REACTION :

Some patients may develop a vasovagal reaction with a sudden drop in blood pressure. It is quite rare with the recumbent position which we follow during sclerotherapy. If the decrease in blood pressure is significant, and sudden, a transient loss of consciousness occurs due to inadequate cerebral perfusion. The treatment involves foot end elevation and for a mo0re severe reaction intravenous administration of atropine 0.5mg is given. If no response is observed, a second dose is given.

POST-SCLEROTHERAPY HYPERPIGMENTATION :

A linear pigmentation along the course of the treated vein can occur within a few weeks. This is attributed to the hemosiderin that is formed out of hemoglobin that has remained in the vein's lumen. It is a part of the resolution process and the patient is to be reassured. This adverse sequel has been described in 30% of the patients treated with sodium –

-tetradecylsulphate [39] Persistent thrombi are thought to produce a subacute perivenulitis that can persist for months. The perivenulitis favours extravasation of RBCs through endothelium or by increasing the permeability of injected endothelium and possibly intratissue fixation of hemosiderin [40], [41]. According to Mitchel. P. Goldman, draining all the thrombi between 2 and 4 weeks of sclerotherapy by puncturing using 23 gauge needle, followed by gentle massage is important in preventing pigmentation. As observed by Carlin MC, Ratz JL the pigmentation lasts from 6 to 24 months [42], which is almost consistent with the results observed in our study (6 to 10 months).

Ecchymosis at and around the injection site may occur due to actinically damaged senescent skin, or due to intake of anticoagulant drugs

TELANGIECTATIC CAPILLARY MATTING :

The appearance of capillary telangiectasias – thread-like, red vessels less than 0.2mm in diameter at and around the treated veins is reported, upto 18%[43] . It is more likely to occur if a higher than necessary concentration of sclerosant is used or if injection pressure is high enough to promote retrograde flow of sclerosant.

INTRAVASCULAR THROMBOSIS :

Treatment associated superficial thrombophlebitis occurs, as immediately after sclerotherapy some amount of blood returns into the lumen even when appropriate compression has been applied. This column of blood gets thrombosed and results in superficial thrombophlebitis. This usually resolves uneventfully within a few weeks or months. The chances of deep vein thrombosis is purely theoretical, as any properly administered sclerosant that has entered the deep venous system will be diluted to an extent as to render it impotent to cause a deep venous mural thrombosis. Post-sclerotherpy compression, initially described by Sigg [44] and Orbach [45] in 1950s and Fegan in 1960 [34], eliminates thrombophlebitic reaction and substitutes a 'sclerophlebitis' with the production of a firm fibrous cord [46].

CUTANEOUS DEVITALIZATION :

An excessive inflammation that develops unexpectedly at a treatment site or a retrograde flow of sclerosant from the treated vein into more proximal capillaries or arterioles at the time of infusion or due to extravasation of the sclerosants [58], [59] . The extravasated sclerosant manifests immediately as a bleb or wheal. At this stage, dessication of the

wound can be prevented by an occlusive ointment. If ulcer develops oral antibiotics and wound dressing along with compression, promotes healing.

ARTERIAL INJECTION :

The most feared complication in sclerotherapy is inadvertent injection of the sclerosant into an artery, though it is very rare [48], [49]. The most common location for this is in the medial malleoli region [50]. The patient will usually, but not always, complains of immediate pain. Cutaneous blanching of the injected site occurs along with loss of pulse and progressive cyanosis of the injected area. This complication is a true ‘sclerotherapy emergency’ and should be immediately treated with 1 ml of procaine 3% as it will complex with STS and renders it inactivate. Immediate heparinization for 6 days with administration of IV dextran 10%, 500ml per day for 3 days is recommended. IV streptokinase may be considered if there are no contraindications. Finally oral prazosin, hydralazine or nifedipine should be considered for 30 days

PULMONARY EMBOLISM AND STOKE :

Fortunately, pulmonary embolism is very rare to occur (1in 40,000). Risk of emboli may be increased by use of estrogens [51]. Most cases have occurred

with high injection volume of about 12ml at a given site. Therefore to minimize this potentially fatal complication, sclerosant quantity should be limited to 0.5 to 1 ml per injection site to prevent leakage of sclerosant into the deep venous system. Rapid compression bandaging of the injection site followed by immediate ambulation or calf movement with frequent ambulation thereafter promotes rapid dilution of sclerosant from the injected site.

- The complications observed in this study were post-sclerotherapy pigmentation (4), edema and pain over injected site(3) and cutaneous ulceration (1)
- Though the above described complications have been reported in various studies, we never experienced any of the serious complications.

DURATION OF VEIN DISAPPEARANCE ;

The duration non resorption of veins depends upon various factors like vein size, volume of blood that has re-entered the lumen after sclerotherapy. Proper compression attempts are required to minimize the volume of blood in the vein. According to Orbach EJ, patients should be examined 2 weeks after injection so that any area of thrombosis can be evacuated early [52]. Each individual area should not be treated sooner than 6

to 8 weeks after injection to allow for adequate healing of the endothelium between treatments.

RECURRENCE AFTER SCLEROTHERAPY :

A great disadvantage of foam injection sclerotherapy is the significant chance of recanalisation of the treated vessels, resulting in recurrence of superficial venous incompetence. Although recurrence is a significant shortcoming of any kind of sclerotherapy, including foam sclerotherapy, it should be remembered that, unlike surgery, foam sclerotherapy is a minimally invasive technique that can be repeated without any real increase in the risks or complexity of the procedure.

POST-SCLEROTHERAPY ACTIVITY :

Immediately after sclerotherapy, patient may participate in all day to day activities.

CONCLUSION

CONCLUSION

Following were the conclusions derived from this study

- Sclerotherapy is a potentially valuable method of treatment for vascular and lymphatic malformations
- Foam sclerotherapy causes maximal sclerosant action at lesser concentration and quantity
- The effect is exerted at microcirculation which cannot be achieved through any other procedures.
- This study demonstrates a complete resolution rate of 89.2% for varicose veins in an average of 4.2 sittings, 70% for hemangiomas – average of 3.9 sittings and 50% in case of lymphangiomas in 5.5 sittings (average), over a period of 18 months.
- The healing of stasis dermatitis (66.7%) and venous ulcers (50%) were found to be accelerated following foam sclerotherapy.
- Though a recurrence rate of 10.7% was noted in patients with varicose veins, foam sclerotherapy is a minimally invasive technique that can be repeated without any real increase in the risks or complexity of the procedure.

- Only minor adverse effects like postsclerotherapy pigmentation , cutaneous devitalisation (1 patient) were noted. No major complications like anaphylaxis, pulmonary embolism or stroke were observed.
- Hence foam sclerotherapy is a simple, safer, feasible, affordable in-office procedure which can be performed without hospitalization or anaesthesia.
- It allows the patient, a rapid return to normal daily activity
- Furthermore it is cheap in comparison to almost all of the other methods available today.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. "Dr. H. I. Biegeleisen, An Expert on Veins, 86". The New York Times. 1991-05-08. http://query.nytimes.com/gst/fullpage.html?res=9D0CE5DC123CF93BA35756C0A967958260. Retrieved 2009-03-05.
2. Goldman M, Sclerotherapy Treatment of varicose and telangiectatic leg vein, Hardcover Text, 2nd Ed, 1995
3. Yamaki T, Nozaki M, Iwasaka S .Comparative study of duplex-guided foam sclerotherapy and duplex-guided liquid sclerotherapy for the treatment of superficial venous insufficiency. Dermatol Surg 2004;30 (5): 718–22
4. Sales CM, Bilof ML, Petrillo KA, Luka NL. Correction of lower extremity deep venous incompetence by ablation of superficial venous reflux. Ann Vasc Surg. Mar 1996;10(2):186-9.
5. Mulliken JB, Glowacki J. Hemangiomas and vascular malformations in infants and children: a classification based on endothelial characteristics. Plast Reconstr Surg. Mar 1982;69(3):412-22.

6. Filston HC. Hemangiomas, cystic hygromas, and teratomas of the head and neck. Semin Pediatr Surg. Aug 1994;3(3):147-59.

7. Blei F, Walter J, Orlow SJ, Marchuk DA. Familial segregation of hemangiomas and vascular malformations as an autosomal dominant trait. Arch Dermatol. Jun 1998;134(6):718-22.

8. Hershkovitz D, Bergman R, Sprecher E. A novel mutation in RASA1 causes capillary malformation and limb enlargement. Arch Dermatol Res. Aug 2008;300(7):385-8.

9. Bielenberg DR, Bucana CD, Sanchez R, et al. Progressive growth of infantile cutaneous hemangiomas is directly correlated with hyperplasia and angiogenesis of adjacent epidermis and inversely correlated with expression of the endogenous angiogenesis inhibitor, IFN-beta. Int J Oncol. Mar 1999;14(3):401-8.

10. Beck L Jr, D'Amore PA. Vascular development: cellular and molecular regulation. FASEB J. Apr 1997;11(5):365-73.

11. Bischoff J. Progenitor cells in infantile hemangioma. J Craniofac Surg. Mar 2009;20 Suppl 1:695-7.

12. Itakura E, Yamamoto H, Oda Y, Furue M, Tsuneyoshi M. VEGF-C and VEGFR-3 in a series of lymphangiomas: Is superficial lymphangioma a true lymphangioma?. Virchows Arch. Mar 2009;454(3):317-25.

13. Finn MC, Glowacki J, Mulliken JB. Congenital vascular lesions: clinical application of a new classification. J Pediatr Surg. Dec 1983;18(6):894-900. [Medline].

14. Hidano A, Nakajima S. Earliest features of the strawberry mark in the newborn. Br J Dermatol. Aug 1972;87(2):138-44.

15. Brown SH Jr, Neerhout RC, Fonkalsrud EW. Prednisone therapy in the management of large hemangiomas in infants and children. Surgery. Feb 1972;71(2):168-73. [Medline].

16. Ezekowitz RA, Mulliken JB, Folkman J. Interferon alfa-2a therapy for life-threatening hemangiomas of infancy. N Engl J Med. May 28 1992;326(22):1456-63.

17. Park CO, Lee MJ, Chung KY. Treatment of unusual vascular lesions: usefulness of sclerotherapy in lymphangioma circumscriptum and acquired digital arteriovenous malformation. Dermatol Surg. Nov 2005;31(11 Pt 1):1451-3.

18. Craig Feied, Sclerosing solutions:The vein book 2007(13):125-131
19. Approved Drug Products with Therapeutic Equivalence equations, Sotradecol ANDA 40-0541 [Nov 12, 2004] US Department of Health and Human services, Food and Drug Administration 2005
20. Tessari L, Cavezzi A, Frullini A. Preliminary experience with a new sclerosing foam in the treatment of varicose veins. Dermatol Surgery 2001;27(1):58-60
21. Winter H, Dräger E, Sterry W. Sclerotherapy for treatment of hemangiomas: Dermatologic Surgery, 2000;28: 11–15.
22. Green D: Sclerotherapy for permanent eradication of varicose veins, J Amer Acad Dermatol 38:461-475, 1998
23. Tollins SH: Treatment of varicose veins- an update, Am J Surg 145:248, 1983
24. Sadick N, Sorhaindo L, Laser Treatment of Telangiectatic and Reticular Veins, Ch 16, p157. The Vein Book / editor, John J. Bergan, 2007.
25. Alessandro Frullini MD, FACP, Attilio Cavezzi MD (2002) Sclerosing Foam in the Treatment of Varicose Veins and Telangiectases: History and Analysis of Safety and Complications. Dermatologic Surgery 28 (1), 11–15

26. Barrett JM, Allen B, Ockelford A, Goldman MP (2004). "Microfoam ultrasound-guided sclerotherapy treatment for varicose veins in a subgroup with diameters at the junction of 10 mm or greater compared with a subgroup of less than 10 mm". Dermatol Surg **30** (11): 1386–90.
27. Kanter A, Thibault P (1996). "Saphenofemoral incompetence treated by ultrasound-guided sclerotherapy". Dermatol Surg **22** (7): 648–52.
28. Padbury A, Benveniste G L, Foam echosclerotherapy of the small saphenous vein, Australian and New Zealand Journal of Phlebology Vol 8, Number 1 (Dec 2004)
29. Breu FX, Guggenbichler S (2004). "European Consensus Meeting on Foam Sclerotherapy, April, 4-6, 2003, Tegernsee, Germany". Dermatol Surg **30** (5): 709–17
30. Breu FX, Guggenbichler S, Wollmann JC (2008). "Duplex ultrasound and efficacy criteria in foam sclerotherapy from the 2nd European Consensus Meeting on Foam Sclerotherapy 2006, Tegernsee, Germany.". Vasa **37** (1): 90–5.
31. Cabrera J, Cabrera Jr A, Garcia-Olmedo MA. Treatment of varicose long saphenous veins with sclerosant in microfoam from:long-term outcomes. Phlebology 2000; **15:**19–23.

32. Fegan WG: Continuous uninterrupted compression technique of varicose veins. Proc R Soc Med 53:837-840, 1960
33. Goldman MP, Bennett RG: Treatment of telangiectsia. A review, J Am Acad Dermatol 17:167, 1987
34. Fegan WG: Continuous compression technique of injecting varicose veins. Lancet 2:109-112, 1963
35. Goldman MP, Kaplan RP, Dufy DM: Postsclerotherapy hyperpigmentation : a histologic evaluation, J Dermtol Surg Oncol 13:547, 1987
36. Duffy DM: Small vessel sclerotherapy: an overview. In Cllen JP, Dahl MV, Golitz LE, et al editors: Advances in dermatology, vol 3, St. Louis, 1988, Mosby.
37. Struckmann K, Christen SJ, Lendorf A, et l: Venous muscle pump improvement by low compression elastic stockings, Phlebology 1:97, 1986
38. Scurr JR, Fisher RK, Wallace SB. Anaphylaxis Following Foam Sclerotherapy: A Life Threatening Complication of Non Invasive Treatment for Varicose Veins. EJVES Extra 2007; 13 (6): 87–89
39. Tournay PR: Sclerosing treatment of very fine intra or subdermal varicosities, Soc Fran de Phlebol 19:235, 1966

40. Manjo G, Palade GE, Schoefl GI: Studies on inflammation: the site of action of histamine and serotonin along the vascular tree: a topographic study, J Biophys Biochem Cytol 11:607, 1961
41. Merlen JF, Coget J, Starteel AM: Pigmentation et stase veineuse, Phlebologie 35:387, 1982
42. Carlin MC, Ratz JL: Treatment of telangiectsia: Comparison of sclerosing agents, J Dermatol Surg Oncol 13:1181, 1987
43. Weiss RA, Weiss MA: Resolution of pain associated with varicose and telangiectatic leg veins after compression sclerotherapy. J Dermatol Surg Oncol 16:333-336, 1990.
44. Sigg K: The treatment of varicosities and accompanying complications, Angiology 3:355, 1952.
45. Orbach EJ: A new approach to the sclerotherapy of varicose veins, Angiology 1:302, 1950.
46. Reid RG, Rothnie NG: Treatment of varicose veins by compression sclerotherapy, Br J Surg 55:889, 1968
47. Campbell B. Varicose veins and their management. BMJ 200; 333 (7562): 287–92.
48. From our legal corresspondent: Hazards of compression sclerotherapy, Br Med J 3:714, 1975.

49. Goldstein M: The complications of sclerotherapy, Phlebology 32:221, 1979.

50. Orbach J: A new look at sclerotherapy, Folia angiologica 25: 181, 1977

51. Fegan WG: The complications of compression sclerotherapy, Practisioner 207:797, 1971

52. Orbach EJ: The importance of removl of post injection coagula during the ourse of sclerotherapy of varicose veins, Vasa 3:475, 1974.

53. Lofgren RA:Varicose veins.their symptoms, complications and mangement, Postgrad Med 65: 131, 1979

54. Beninson J, Livingood CS: Stasis dermatitis in clinical dermatology, In Dermis DJ, editor: clinical dermatology, vol 2, Philadelphia, 1985, Harper & Row

55. Baron HC: Varicose veins, Consultant May 1983, p.108

56. Lindemyr H, Santler R: The fibrinolytic activity of the vein wall, Phlebologie 30:151, 1977

57. Modified from Duffy DM: Small vessel sclerotherapy: an overview. In Callen JP, Dahl MV, Golitz LE, et al, editors: Advances in dermatology, vol 3, St Louis. 1988, Mosby

58. Tretbar LL:Spider angiomata. Treatment with sclerosing injections, J Kansas Med Soc 79:198, 1978

59. Mantse L:A mild sclerosing agent for telangiectasias, J Dermatol Surg Oncol 11:9, 1985

ANNEXURES

PROFORMA

Name Age Sex

Skin O.P No :

Occupation :

Address :

Per capita income :

Presenting complaints :

H/o present illness :

onset

duration

course

H/o associated symptoms

H/o leg ulcers

H/o predisposing factors

H/o aggravating / relieving factors

H/o abdominal distension/ mass/ pain

H/o other systemic disturbances

Past history :

H/o any surgeries for varicose veins / hemangiomas in the past

H/o any abdominal surgeries

Family history :

Personal history :

Diet :

Sleep :

Appetite :

Bowel & bladder habits :

Habits : smoking / alcoholism

Menstrual history (in females):

Menarche/menopause; cycles; flow

Obstetric history (in females):

H/o pregnancy / lactation

H/o intake of OCPs

Treatment history :

H/o previous treatment / surgeries

H/o allergy to sclerosants

General Physical Examination :

Consciousness; orientation

Built & nourishment

Febrile/afebrile

Pallor / cyanosis / clubbing / icterus / lymphadenopathy / pedal edema

Pulse rate : Peripheral pules :

Blood pressure :

Respiratory rate ;

Temperature :

Systemic Examination :

Cardiovascular system :

Respiratory system :

Abdomen (including genital and per rectal examination):

Central nervous system :

Local Examination :

I Hemangiomas:

Inspection:

- Number ; size ; site ; shape ; surface ; skin around the swelling ;

cough impulse

Palpation :

- extent ; warmth ; tenderness ; pulsation/thrill ; compressibility; cough impulse ; girth of the limb

Auscultation :

- bruit +/-

II Varicose veins :

Inspection :

- site & extent of varicosity
- surrounding skin

Palpation :

- warmth
- tenderness
- pitting edema of leg

CLINICAL TESTS:

- Tourniquet test

A tourniquet is applied just below the level of the SFJ and the patient is then asked to stand. Rapid filling of the varicosities with the tourniquet still on suggests incompetent perforators below the level of the SFJ. If no

filling is seen at this point, the tourniquet is released. Rapid filling of the varicosities at this stage suggests SFJ incompetence.

- Brodie-Trendelenberg test

Similar to tourniquet with compression given at the saphenofemoral junction using thumb.

- Fegan's test

On standing, the site where the perforators enter the deep fascia bulges and this is marked. On lying down, button like depressions in the deep fascia are felt at the marked out points which confirms the site of perforators.

- Pratt's test

An Esmarch bandage is applied to the leg from below upwards with a tourniquet at the saphenofemoral junction. After the bandage is released, perforators are seen as 'blow-outs'

- Perthes test

The affected limb is wrapped with elastic bandage, and the patient is asked to walk around and exercise. Development of severe pain indicates DVT.

- Homan’s sign

Dorsiflexion of foot elicits pain in posterior calf.

- Moses sign

Squeezing of posterior calf elicits pain.

Percussion :

- Schwartz’s test

This technique is useful in determining whether 2 venous segments are directly interconnected. With the patient in a standing position, a vein segment is percussed at one position while an examining hand feels for a "pulse wave" at another position

Auscultation :

- Any continuous machinery murmur of AV fistula +/-

Examination of ulcer (if any)

Examination of skin changes (if any**)**

DIAGNOSIS :

INVESTIGATIONS :

Hb%, TC, DC, ESR, Platelet

Urine – albumin, sugar, deposits

BT, CT

Doppler ultrasonogram both limbs

Ultrasonogram abdomen & pelvis

CT scan (as required)

MRI (as required)

MASTER CHART – VARICOSE VEINS

S.No	NAME	AGE	SEX	SYMPTOM	VESSEL TYPE	BT	CT	STS CONC. (%)	NO.OF SITTINGS	SYMPTOM AFTER TRT	RESOLUTION
1.	MURALI	35	M	Stasis eczema	4	2’10”	4’11”	1	4	Reduced	Complete
2.	GOPALAN	45	M	Pain	1A	3’12”	5’21”	1	5	Persistent	Complete
3.	DAYALAN	38	M	Discolouration	3	4’11”	4’9”	0.1	2	Persistent	Complete
4.	VEERAN	55	M	Stasis eczema	2	2’9”	4’14”	0.75	3	Persistent	Complete
5.	SUMATHI	34	F	Itching	4	2’14”	6’16”	1.5	5	Reduced	Complete
6.	PANDIYAN	48	M	Stasis eczema	4	2’6”	5’10”	2	4	Reduced	Complete
7.	GODHANDAM	37	M	Leg swelling	4	2’10”	4’16”	1	6	Reduced	Complete
8.	LAKSHMI	38	F	Leg ulcer	4	3’10”	6’12”	1.5	5	Healed	Complete
9.	KANNAN	50	M	Stasis eczema	4	2’8”	5’8”	0.25	2	Persistent	Complete
10.	SIVARAMAN	32	M	Leg ulcer	2	2’10”	4’30”	1	4	Reduced	Complete
11.	PARAMASIVAM	42	M	Stasis eczema	4	4’00”	7’11”	1.5	6	Reduced	Complete
12.	SHANTHI	48	F	Stasis eczema	4	2’13”	4’18”	1	4	Reduced	Complete
13.	RIYAZ	53	M	Stasis eczema	1B	2’8”	5’11”	1.5	5	Reduced	Recurrence
14.	RAHIMA	35	F	Pain	2	3’13”	4’10”	0.75	3	Reduced	Complete
15.	RAMESH	44	M	Stasis eczema	3	2’14”	4’15”	1.5	5	Reduced	Complete
16.	SUBBAMMAL	36	F	Discolouration	4	2’16”	6’14”	1	4	Persistent	Complete
17.	VELAN	40	M	Itching	4	2’5”	5’10”	0.5	2	Reduced	Complete
18.	SUBRAMANI	39	M	Stasis eczema	4	2’10”	5’18”	1	6	Persistent	Complete
19.	MANIKANDAN	43	M	Stasis eczema	4	2’17”	4’40”	1.5	4	Reduced	Complete
20.	GANDHI	33	M	Leg ulcer	3	2’15”	5’6”	1	5	Persistent	Complete
21.	ESWARAN	47	M	Discolouration	4	3’10”	5’12”	1.5	6	Reduced	Complete
22.	MADHIVANAN	37	M	Pain	4	3’15”	6’11”	0.75	3	Reduced	Complete
23.	RAJARAMAN	32	M	Stasis eczema	2	3’30”	7’00”	0.5	2	Persistent	Complete
24.	KUMAR	43	M	Leg swelling	4	4’10”	4’11”	0.75	5	Reduced	Recurrence
25.	PRAMILA	35	F	Leg ulcer	4	3’12”	4’00”	0.75	3	Healed	Complete
26.	SENTHIL	45	M	Stasis eczema	4	5’10”	6’18”	1	4	Reduced	Complete
27.	SARAVANAN	43	M	Leg swelling	4	2’10”	4’11”	6	6	Reduced	Complete
28.	IYYANAR	50	M	Discolouration	4	3’20”	5’10”	1	4	Persistent	Recurrence

ABBREVIATIONS : BT – Bleeding time; CT –Clotting time; STS – Sodium tetradecyl sulphate; conc. - concentration

MASTER CHART – HEMANGIOMA & LYMPHANGIOMA

S.No	NAME	AGE	SEX	SITE	BT	CT	STS CONC. (%)	NO.OF SITTINGS	RESOLUTION
HEMANGIOMA									
1.	RAGHU	23	M	Gluteal	3’10”	5’10”	O.75	6	Near complete
2.	SAVITHA	16	F	Trunk	2’18”	6’18”	O.75	4	Complete
3.	JAYARAJ	19	M	Tongue	2’10”	4’40”	O.75	3	Complete
4.	RAVI	24	M	leg	4’20”	4’6”	O.75	5	Near complete
5.	JAGAN	20	M	Forearm	2’13”	5’12”	O.75	3	Complete
6.	MARY	17	F	Hand	2’8”	7’10”	O.75	3	Complete
7.	SATHISH	14	M	Face	3’13”	3’18”	O.75	2	Complete
8.	JEBARAJ	30	M	Trunk	4’10”	4’40”	O.75	5	Near complete
9.	SATHYA	27	F	Tongue	2’8”	4’6”	O.75	6	Complete
10.	HUSSAIN	31	M	Hand	3’10”	3’12”	O.75	2	Complete
LYMPHANGIOMA									
1.	SELVI	14	F	Tongue	3’13”	5’18”	O.75	6	Complete
2.	MANOJ	22	M	Gluteal	4’20”	4’20”	O.75	5	Near complete

ABBREVIATIONS : BT – Bleeding time; CT –Clotting time; STS – Sodium tetradecyl sulphate; conc. - concentration